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TO: Alton Pryor Location: Art Unit: 1616 August 18, 2004 Case Serial Number: 09/328742	From: P. Sheppard Location: Remsen Building Phone: (571) 272-2529 sheppard@uspto.gov

Search Notes

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=> fil hcaplus
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FILE COVERS 1907 - 18 Aug 2004  VOL 141 ISS 8
FILE LAST UPDATED: 17 Aug 2004  (20040817/ED)

 This file contains CAS Registry Numbers for easy and accurate
 substance identification.


=>
=>

=> d stat que
L1              STR

C≡≡C
1   2


NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS   2

STEREO ATTRIBUTES: NONE
L2              SCR 2039 OR 2041 OR 2050 OR 2049 OR 2048 OR 2053 OR 2052 O
R 2051 OR 2043
L3              STR

             6
             O
             ‖
C~^G1^^^^^^-C^^^N
1   2        4   5


REP G1=(10-20) C
NODE ATTRIBUTES:
NSPEC   IS RC     AT   5
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS  5
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STEREO ATTRIBUTES: NONE
L4 12044 SEA FILE=REGISTRY SSS FUL (L3 AND L1) NOT L2
L5 STR

NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 2

STEREO ATTRIBUTES: NONE
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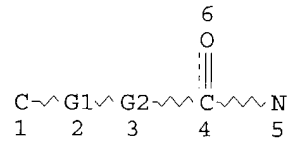
L7 STR



REP G1=(20-20) C
REP G2=(1-10) C
NODE ATTRIBUTES:
NSPEC IS RC AT 5
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 6

STEREO ATTRIBUTES: NONE
L8 1164 SEA FILE=REGISTRY SSS FUL (L7 AND L5) NOT L6
L9 STR

NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 2

STEREO ATTRIBUTES: NONE
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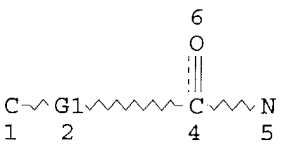
R 2051 OR 2043
L11 STR



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NODE ATTRIBUTES:
NSPEC IS RC AT 5
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 5

STEREO ATTRIBUTES: NONE
L12 25296 SEA FILE=REGISTRY SSS FUL (L11 AND L9) NOT L10
L13 STR

NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 2

STEREO ATTRIBUTES: NONE
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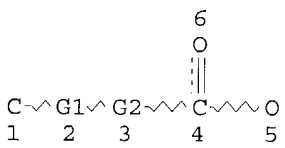
R 2051 OR 2043 OR 1838
L15 STR



REP G1=(3-20) C
REP G2=(0-10) C
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 6

STEREO ATTRIBUTES: NONE
L16 73850 SEA FILE=REGISTRY SSS FUL (L15 AND L13) NOT L14
L17 STR

NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 2

STEREO ATTRIBUTES: NONE
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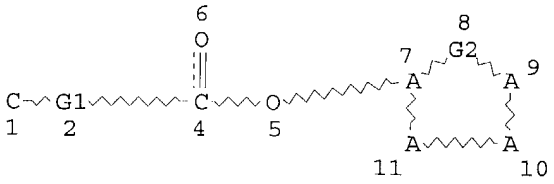
R 2051 OR 2043 OR 1840
L19 STR



REP G1=(5-9) C
REP G2=(0-4) A
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RSPEC I
NUMBER OF NODES IS 10

STEREO ATTRIBUTES: NONE
L20 2370 SEA FILE=REGISTRY SSS FUL (L19 AND L17) NOT L18
L21 STR

NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 2

STEREO ATTRIBUTES: NONE
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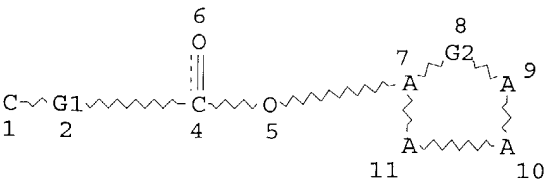
R 2051 OR 2043 OR 1840
L23 STR

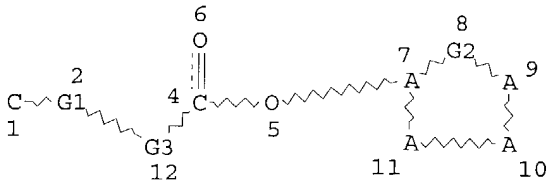


REP G1=(10-20) C
REP G2=(0-4) A
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RSPEC I
NUMBER OF NODES IS 10

STEREO ATTRIBUTES: NONE

L24 STR



REP G1=(20-20) C
REP G2=(0-4) A
REP G3=(1-10) C
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

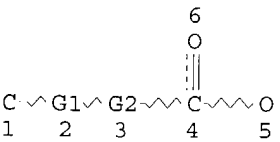
GRAPH ATTRIBUTES:
RSPEC I
NUMBER OF NODES IS 11

STEREO ATTRIBUTES: NONE
L25 1058 SEA FILE=REGISTRY SSS FUL ((L24 OR L23) AND L21) NOT L22
L26 STR

NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 2

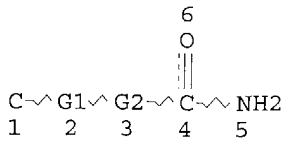
STEREO ATTRIBUTES: NONE
L27 SCR 2039 OR 2041 OR 2050 OR 2049 OR 2048 OR 2053 OR 2052 O
R 2051 OR 2043
L28 STR



REP G1=(3-20) C
REP G2=(0-10) C
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 6

STEREO ATTRIBUTES: NONE
L29 13336 SEA FILE=REGISTRY SSS FUL (L28 AND L26) NOT L27
L30 108806 SEA FILE=REGISTRY ABB=ON PLU=ON L4 OR L8 OR L12 OR L16 OR
L20 OR L25 OR L29
L31 STR



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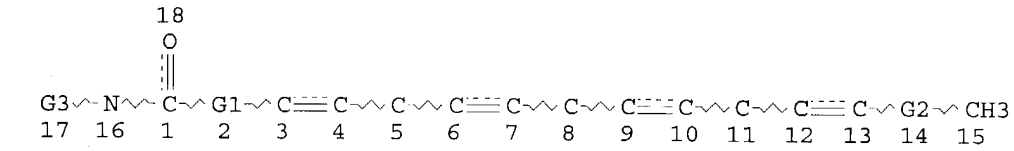
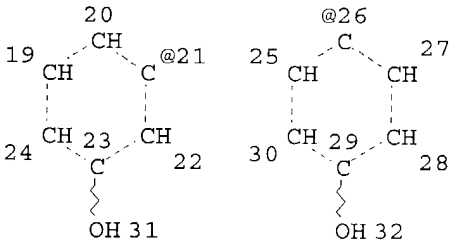
REP G2=(0-1) C
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 6

STEREO ATTRIBUTES: NONE
L33 STR





REP G1=(3-3) C
REP G2=(4-4) C
VAR G3=21/26
NODE ATTRIBUTES:
DEFAULT MLEVEL IS ATOM
DEFAULT ECLEVEL IS LIMITED

GRAPH ATTRIBUTES:
RING(S) ARE ISOLATED OR EMBEDDED
NUMBER OF NODES IS 32

STEREO ATTRIBUTES: NONE

L34	83	SEA FILE=REGISTRY SUB=L30 SSS FUL L31 OR L33
L35	108723	SEA FILE=REGISTRY ABB=ON PLU=ON L30 NOT L34
L36	5	SEA FILE=REGISTRY ABB=ON PLU=ON ANANDAMIDE
L38	186720	SEA FILE=HCAPLUS ABB=ON PLU=ON L35
L39	1358	SEA FILE=HCAPLUS ABB=ON PLU=ON L36 OR ?ANANDAMID?
L43	70	SEA FILE=HCAPLUS ABB=ON PLU=ON INHIBIT?(L)TRANSPORT(L)L39
L44	62	SEA FILE=HCAPLUS ABB=ON PLU=ON L43 AND L38
L45	9	SEA FILE=HCAPLUS ABB=ON PLU=ON L44 AND PD=<JUNE 9, 1999

=>
=>

=> d ibib abs hitstr l45 1-9

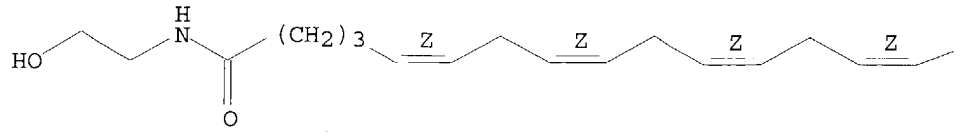
L45 ANSWER 1 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:510255 HCAPLUS
DOCUMENT NUMBER: 131:295096
TITLE: Unsaturated Long-Chain N-Acyl-vanillyl-amides (N-AVAMs): Vanilloid Receptor Ligands That **Inhibit Anandamide**-Facilitated **Transport** and Bind to CB1 Cannabinoid Receptors
AUTHOR(S): Melck, Dominique; Bisogno, Tiziana; De Petrocellis, Luciano; Chuang, Huai-hu; Julius, David; Bifulco, Maurizio; Di Marzo, Vincenzo
CORPORATE SOURCE: Istituto per la Chimica di Molecole di Interesse Biologico, Consiglio Naz. Ric., Arco Felice, Napoli, 80072, Italy
SOURCE: Biochemical and Biophysical Research Communications (**1999**), 262(1), 275-284
CODEN: BBRCA9; ISSN: 0006-291X
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We investigated the effect of changing the length and degree of unsatn. of the fatty acyl chain of N-(3-methoxy-4-hydroxy)-benzyl-cis-9-octadecenoamide (olvanil), a ligand of vanilloid receptors, on its capability to: (i) **inhibit anandamide**-facilitated **transport** into cells and enzymic hydrolysis, (ii) bind to CB1 and CB2 cannabinoid receptors, and (iii) activate the VR1 vanilloid receptor. Potent **inhibition** of [14C]**anandamide** accumulation into cells was achieved with C20:4 n-6, C18:3 n-6 and n-3, and C18:2 n-6 N-acyl-vanillyl-amides (N-AVAMs). The saturated analogs and Δ9-trans-olvanil were inactive. Activity in CB1 binding assays increased when increasing the number of cis-double bonds in a n-6 fatty acyl chain and, in saturated N-AVAMs, was not greatly sensitive to decreasing the chain length. The C20:4 n-6 analog (arvanil) was a potent **inhibitor** of **anandamide** accumulation (IC50 = 3.6 μM) and was 4-fold more potent than **anandamide** on CB1 receptors (Ki = 0.25-0.52 μM), whereas the C18:3 n-3 N-AVAM was more selective than arvanil for the uptake (IC50 = 8.0 μM) vs. CB1 receptors (Ki = 3.4 μM). None of the compds. efficiently **inhibited** [14C] **anandamide** hydrolysis or bound to CB2 receptors. All N-AVAMs activated the cation currents coupled to VR1 receptors overexpressed in Xenopus oocytes. In a simple, intact cell model of both vanilloid- and **anandamide**-like activity, i.e., the **inhibition** of human breast cancer cell (HBCC) proliferation, arvanil was shown to behave as a "hybrid" activator of cannabinoid and vanilloid receptors. (c) 1999 Academic Press.

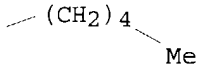
IT **94421-68-8, Anandamide**
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(unsatd. long-chain N-acyl-vanillyl-amides as vanilloid receptor ligands that **inhibit anandamide**-facilitated **transport** and bind to CB1 cannabinoid receptors)
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



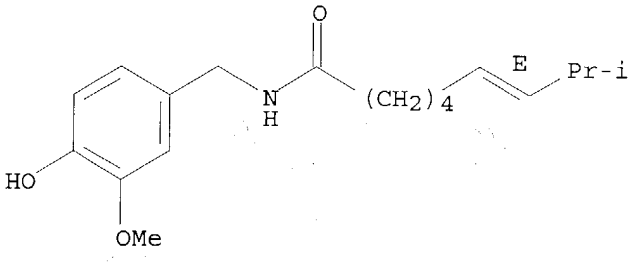
PAGE 1-B



IT **404-86-4**, Capsaicin **16729-47-8 58493-49-5**,
Olvanil **95548-23-5 104899-01-6 104926-32-1**
128007-31-8
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(unsatd. long-chain N-acyl-vanillyl-amides as vanilloid receptor ligands that **inhibit anandamide**-facilitated **transport** and bind to CB1 cannabinoid receptors)

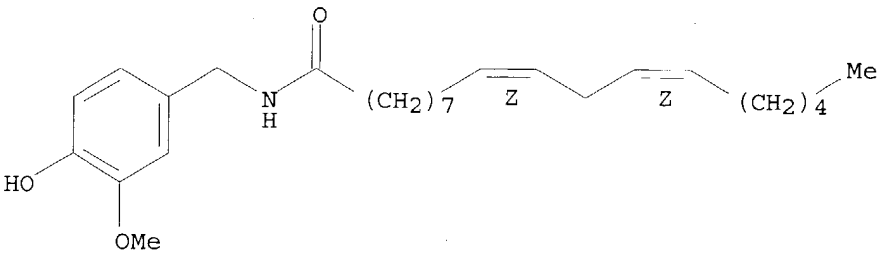
RN 404-86-4 HCAPLUS
CN 6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 16729-47-8 HCAPLUS
CN 9,12-Octadecadienamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z,12Z)- (9CI) (CA INDEX NAME)

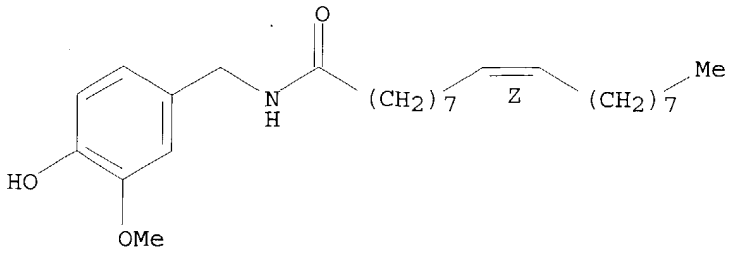
Double bond geometry as shown.



RN 58493-49-5 HCAPLUS
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z)- (9CI) (CA

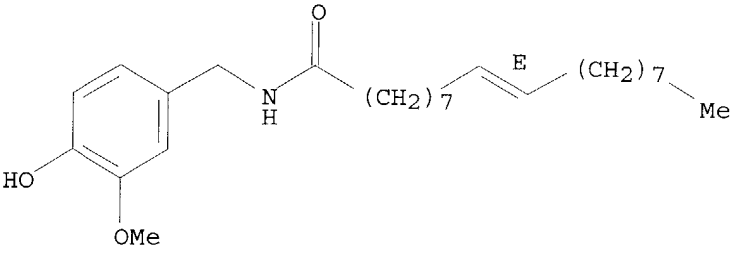
INDEX NAME)

Double bond geometry as shown.



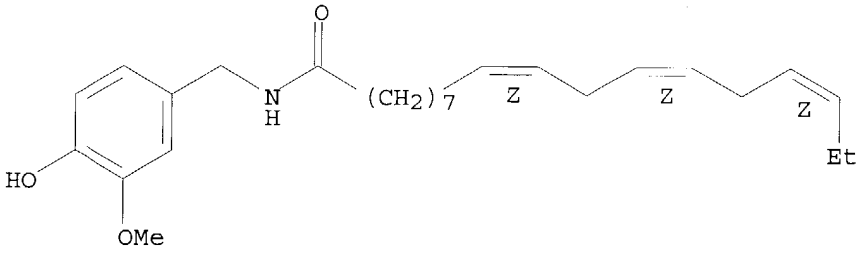
RN 95548-23-5 HCAPLUS
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



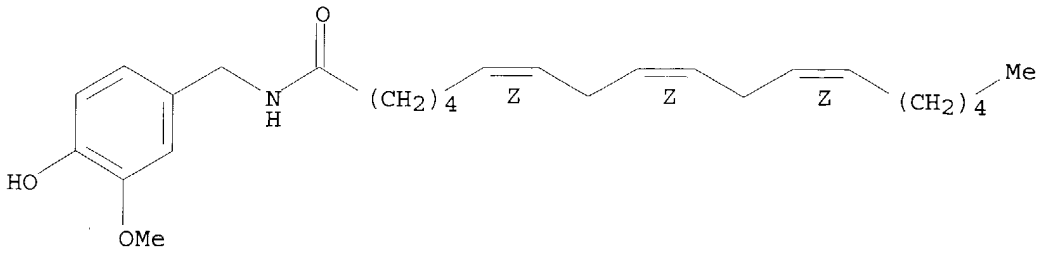
RN 104899-01-6 HCAPLUS
CN 9,12,15-Octadecatrienamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 104926-32-1 HCAPLUS
CN 6,9,12-Octadecatrienamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (6Z,9Z,12Z)- (9CI) (CA INDEX NAME)

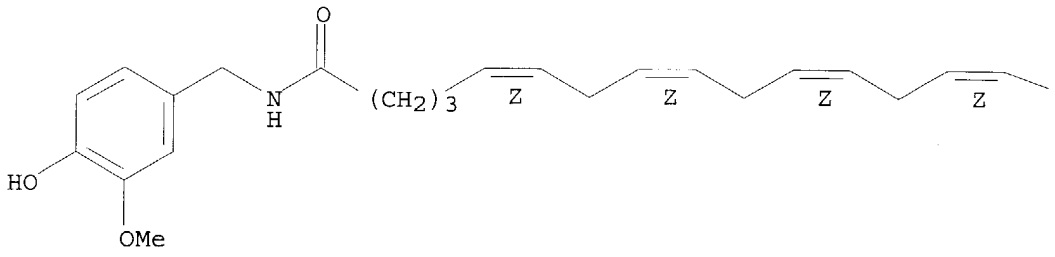
Double bond geometry as shown.



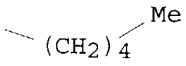
RN 128007-31-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



REFERENCE COUNT: 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 2 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:477642 HCAPLUS
DOCUMENT NUMBER: 131:251951
TITLE: Structure-activity relationships of anandamide, an endogenous cannabinoid ligand
AUTHOR(S): Khanolkar, Atmaram D.; Makriyannis, Alexandros
CORPORATE SOURCE: Departments of Pharmaceutical Sciences, University of Connecticut, Storrs, CT, 06269, USA
SOURCE: Life Sciences (**1999**), 65(6/7), 607-616
CODEN: LIFSAK; ISSN: 0024-3205
PUBLISHER: Elsevier Science Inc.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 45 refs. Identification of arachidonylethanolamide (**anandamide**) as an endogenous cannabinoid is one of the most important developments in cannabinoid research in recent years. In a relatively short period of time thereafter, pharmacol. and biochem. studies have confirmed initial speculations that **anandamide** is a neuromodulator and significantly advanced our understanding of cannabinoid biochem. Moreover, the discovery of **anandamide** has led to the

identification of two heretofore unknown proteins associated with cannabinoid physiol.: (1)**Anandamide** Amidohydrolase (AAH), an enzyme responsible for the hydrolytic breakdown of **anandamide** and (2) the **Anandamide** Transporter (ANT), a carrier protein involved in the **transport** of **anandamide** across the cell membrane. Evidence obtained so far suggests that these two proteins, in combination, are responsible for the termination of the biol. actions of **anandamide.** Also, the discovery of **anandamide** has revealed a novel class of more selective cannabimimetic agents possessing a somewhat different pharmacol. profile of potential therapeutic value. A number of such analogs have now been reported many of which possess markedly improved cannabinoid receptor affinity and metabolic stability compared to those of the parent ligand. Generally, **anandamide** and all known analogs exhibit significant selectivity for the CB1 receptor and modest to very low affinity for CB2. For this reason, this group of compds. can be considered as CB1 ligands. The purpose of this review is to summarize the structure-activity relationships (SAR) of **anandamide** for the CB1 cannabinoid receptor and to define the structural requirements for the substrates and the **inhibitors** of **anandamide** amidohydrolase and the **anandamide** transporter.

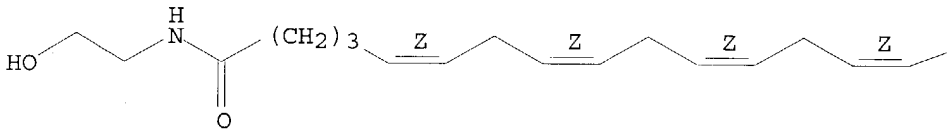
IT **94421-68-8**, Anandamide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(structure-activity relationships of anandamide, endogenous cannabinoid ligand)

RN 94421-68-8 HCAPLUS

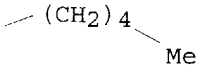
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 3 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:314488 HCAPLUS
DOCUMENT NUMBER: 131:100242
TITLE: Structural determinants for recognition and translocation by the anandamide transporter
AUTHOR(S): Piomelli, D.; Beltramo, M.; Glasnapp, S.; Lin, S. Y.; Goutopoulos, A.; Xie, Xiang-Qun; Makriyannis, A.
CORPORATE SOURCE: The Neurosciences Institute, San Diego, CA, 92121, USA
SOURCE: Proceedings of the National Academy of Sciences of the United States of America (**1999**), 96(10), 5802-5807
CODEN: PNASA6; ISSN: 0027-8424

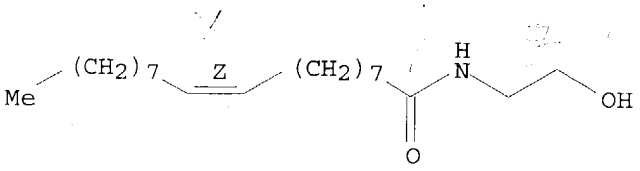
PUBLISHER: National Academy of Sciences
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The biol. actions of **anandamide** (arachidonylethanolamide), an endogenous cannabinoid lipid, are terminated by a two-step inactivation process consisting of carrier-mediated uptake and intracellular hydrolysis. **Anandamide** uptake in neurons and astrocytes is mediated by a high-affinity, Na+-independent transporter that is selectively **inhibited** by N-(4-hydroxyphenyl)-arachidonamide (AM404). In the present study, we examined the structural determinants governing recognition and translocation of substrates by the **anandamide** transporter constitutively expressed in a human astrocytoma cell line. Competition expts. with a select group of analogs suggest that substrate recognition by the transporter is favored by a polar nonionizable head group of defined stereochem. configuration containing a hydroxyl moiety at its distal end. The secondary carboxamide group interacts favorably with the transporter, but may be replaced with either a tertiary amide or an ester, suggesting that it may serve as hydrogen acceptor. Thus, 2-arachidonylglycerol, a putative endogenous cannabinoid ester, also may serve as a substrate for the transporter. Substrate recognition requires the presence of at least one cis double bond situated at the middle of the fatty acid carbon chain, indicating a preference for ligands whose hydrophobic tail can adopt a bent U-shaped conformation. On the other hand, uptake expts. with radioactively labeled substrates show that no fewer than four cis nonconjugated double bonds are required for optimal translocation across the cell membrane, suggesting that substrates are transported in a folded hairpin conformation. These results outline the general structural requisites for **anandamide transport** and may assist in the development of selective **inhibitors** with potential clin. applications.

IT **111-58-0 506-32-1 1808-26-0 2566-89-4**
24257-12-3 35474-99-8 53847-30-6
94421-68-8 150314-34-4 156910-28-0
157182-49-5 157182-50-8 162758-93-2
162758-96-5 164228-51-7 166100-34-1
183718-67-4 187224-16-4 187224-18-6
213027-54-4 231632-70-5 231632-71-6
231632-72-7 231632-73-8 231632-74-9
231632-75-0 231632-76-1 231632-77-2
RL: BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); PROC (Process)
(structural determinants for recognition and translocation by the anandamide transporter)

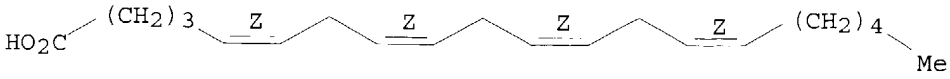
RN 111-58-0 HCAPLUS
CN 9-Octadecenamide, N-(2-hydroxyethyl)-, (9Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



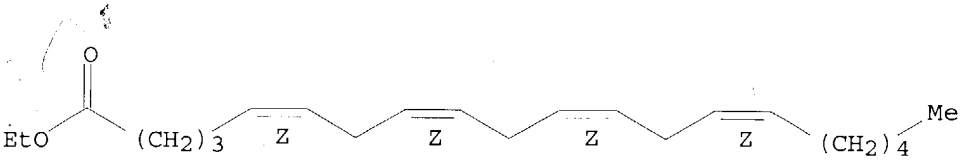
RN 506-32-1 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



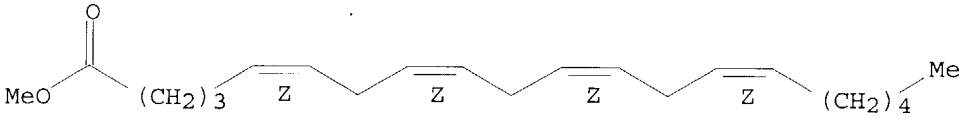
RN 1808-26-0 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, ethyl ester, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



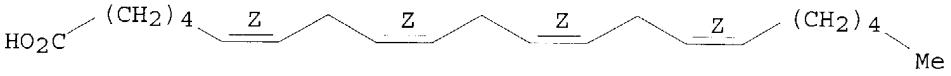
RN 2566-89-4 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, methyl ester, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 24257-12-3 HCAPLUS
CN 6,9,12,15-Heneicosatetraenoic acid, (6Z,9Z,12Z,15Z)- (9CI) (CA INDEX NAME)

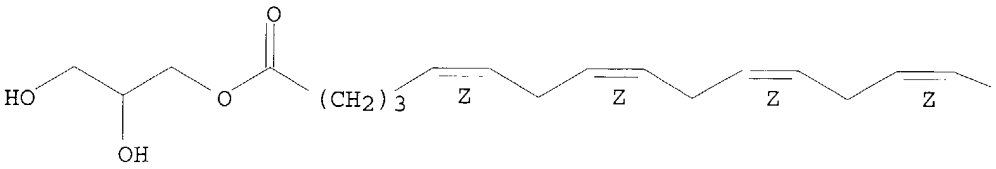
Double bond geometry as shown.



RN 35474-99-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, 2,3-dihydroxypropyl ester, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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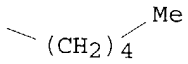
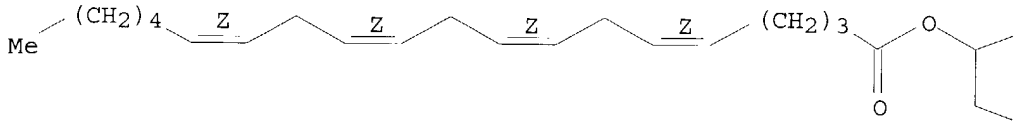


RN 53847-30-6 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, 2-hydroxy-1-(hydroxymethyl)ethyl ester, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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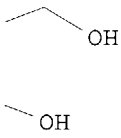
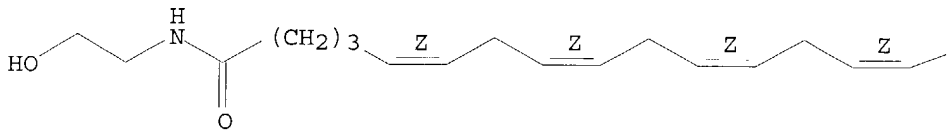


RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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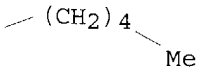
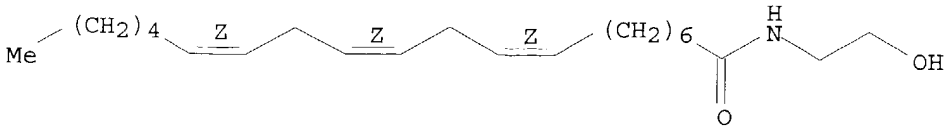


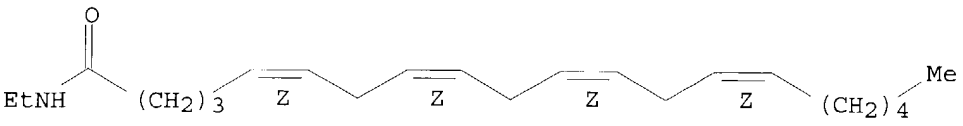
RN 150314-34-4 HCAPLUS
CN 8,11,14-Eicosatrienamide, N-(2-hydroxyethyl)-, (8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 156910-28-0 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-ethyl-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

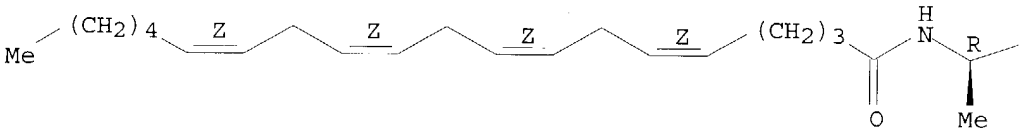
Double bond geometry as shown.



RN 157182-49-5 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-[(1R)-2-hydroxy-1-methylethyl]-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).
Double bond geometry as shown.

PAGE 1-A



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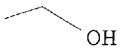
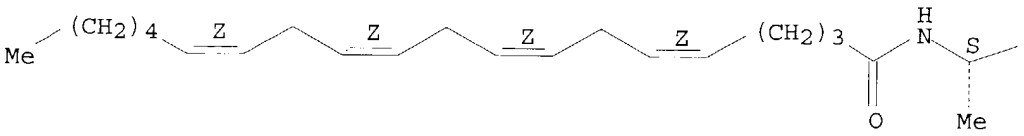


RN 157182-50-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-[(1S)-2-hydroxy-1-methylethyl]-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Absolute stereochemistry.
Double bond geometry as shown.

PAGE 1-A



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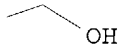
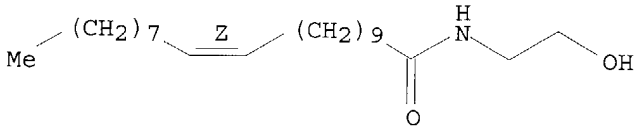


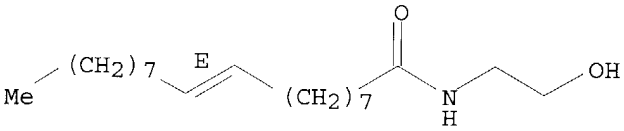
RN 162758-93-2 HCAPLUS
CN 11-Eicosenamide, N-(2-hydroxyethyl)-, (11Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 162758-96-5 HCAPLUS
CN 9-Octadecenamide, N-(2-hydroxyethyl)-, (9E)- (9CI) (CA INDEX NAME)

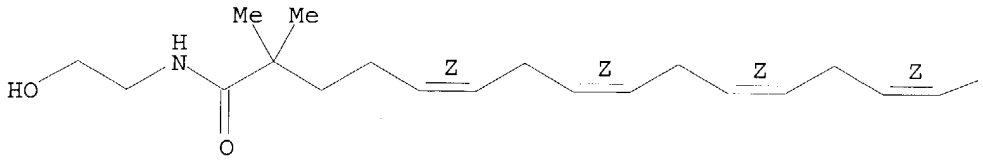
Double bond geometry as shown.



RN 164228-51-7 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-2,2-dimethyl-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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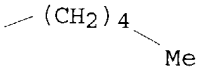
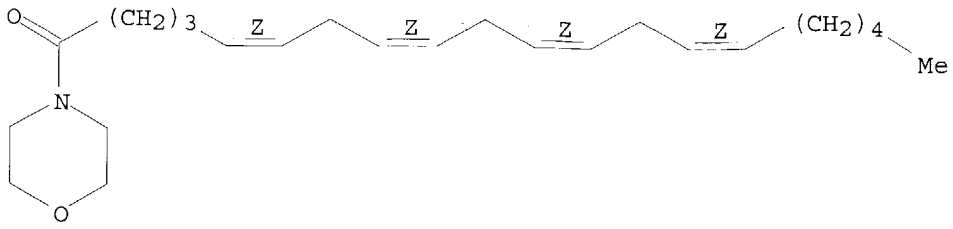


RN 166100-34-1 HCAPLUS
CN Morpholine, 4-[(5Z,8Z,11Z,14Z)-1-oxo-5,8,11,14-eicosatetraenyl]- (9CI) (CA INDEX NAME)

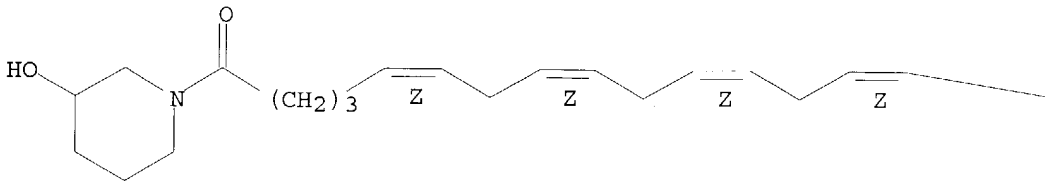
Double bond geometry as shown.



RN 183718-67-4 HCAPLUS
CN 3-Piperidinol, 1-[(5Z,8Z,11Z,14Z)-1-oxo-5,8,11,14-eicosatetraenyl]- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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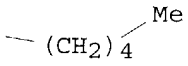
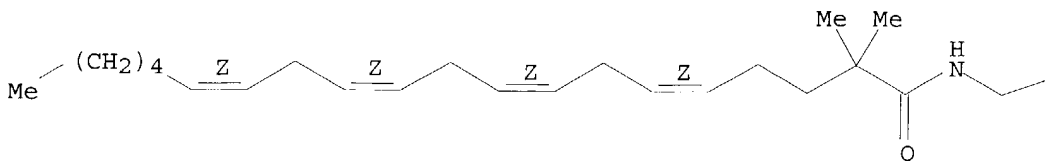


RN 187224-16-4 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-[(2S)-2-hydroxypropyl]-2,2-dimethyl-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (+).
Double bond geometry as shown.

PAGE 1-A



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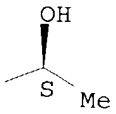
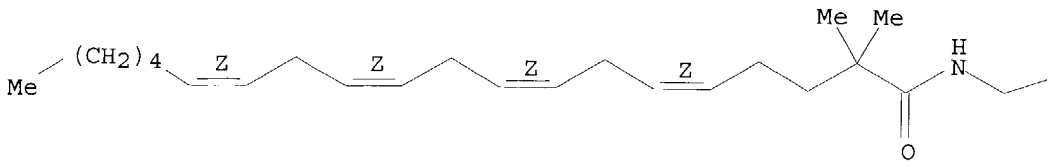


RN 187224-18-6 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-[(2R)-2-hydroxypropyl]-2,2-dimethyl-,

(5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Absolute stereochemistry. Rotation (-).
Double bond geometry as shown.

PAGE 1-A



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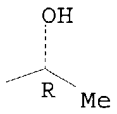
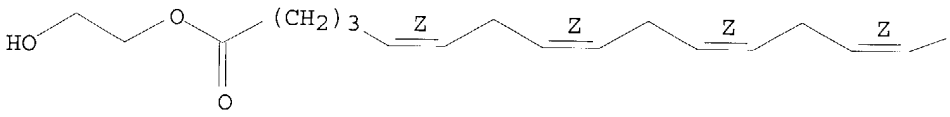


RN 213027-54-4 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, 2-hydroxyethyl ester, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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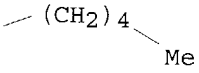
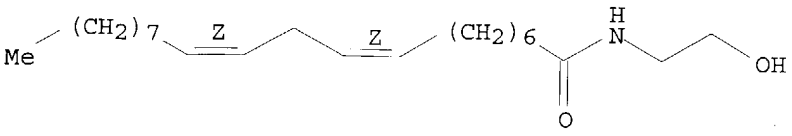
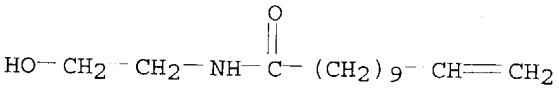


RN 231632-70-5 HCAPLUS
CN 8,11-Eicosadienamide, N-(2-hydroxyethyl)-, (8Z,11Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



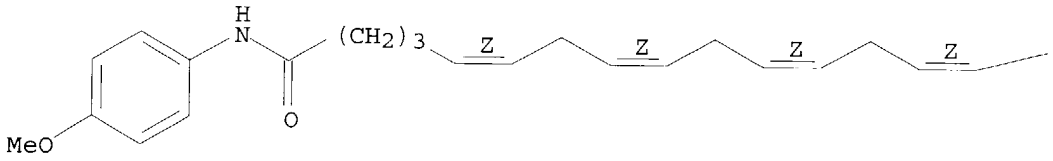
RN 231632-71-6 HCAPLUS
CN 11-Dodecenamide, N-(2-hydroxyethyl)- (9CI) (CA INDEX NAME)



RN 231632-72-7 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(4-methoxyphenyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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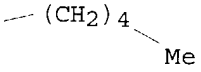
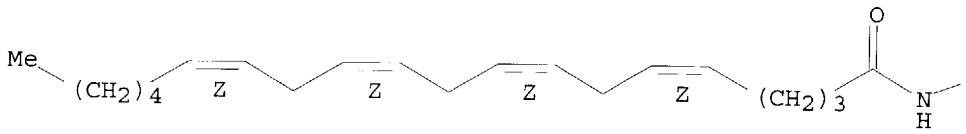


RN 231632-73-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(4-methylphenyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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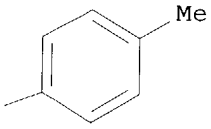
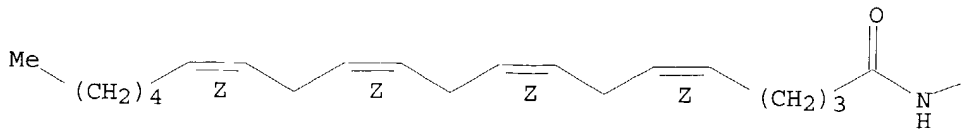


RN 231632-74-9 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(4-cyanophenyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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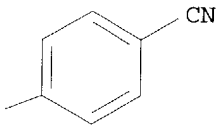
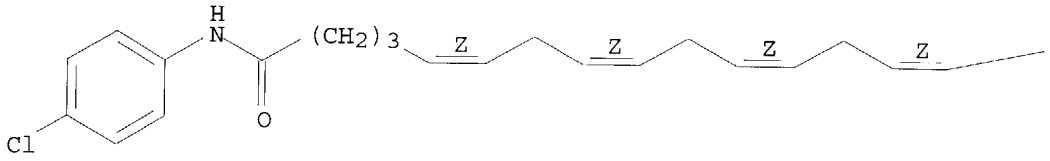


RN 231632-75-0 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(4-chlorophenyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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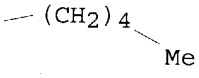
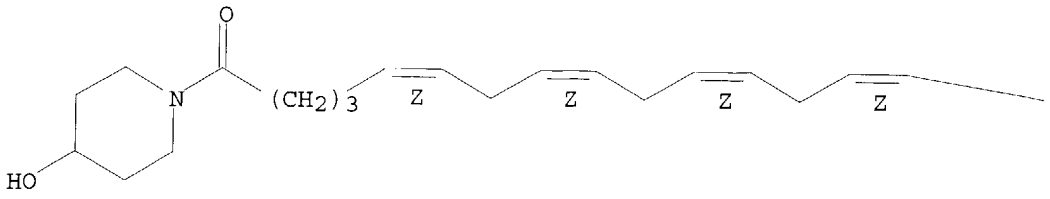
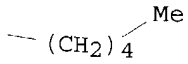


RN 231632-76-1 HCAPLUS
CN 4-Piperidinol, 1-[(5Z,8Z,11Z,14Z)-1-oxo-5,8,11,14-eicosatetraenyl]- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

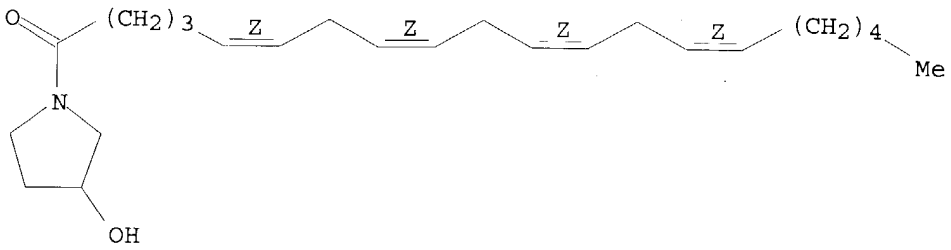
PAGE 1-A





RN 231632-77-2 HCAPLUS
CN 3-Pyrrolidinol, 1-[(5Z,8Z,11Z,14Z)-1-oxo-5,8,11,14-eicosatetraenyl]- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

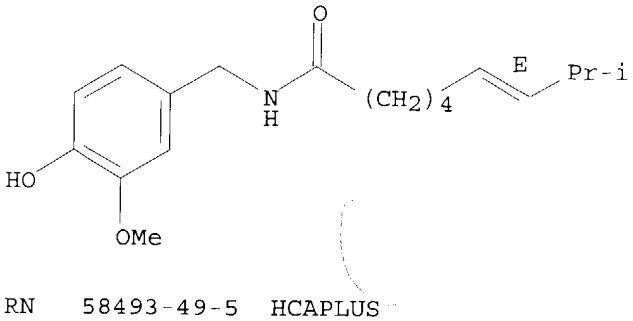
L45 ANSWER 4 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:811082 HCAPLUS
DOCUMENT NUMBER: 130:218085
TITLE: **Anandamide transport inhibition** by the vanilloid agonist olvanil
AUTHOR(S): Beltramo, Massimiliano; Piomelli, Daniele
CORPORATE SOURCE: The Neurosciences Institute, San Diego, CA, 92121, USA
SOURCE: European Journal of Pharmacology (**1999**), 364(1), 75-78
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The structural similarities between the **anandamide transport inhibitor** N-(4-hydroxyphenyl)-arachidonylamide (AM404) and the synthetic vanilloid agonist olvanil [(N-vanillyl)-9-oleamide], prompted us to investigate the possibility that olvanil may interfere with **anandamide transport.** The intracellular accumulation of [3H]**anandamide** by human astrocytoma cells was prevented by olvanil with a Ki value of 14.1±7.1 μM. By contrast, capsaicin [(8-methyl-N-vanillyl)-6-noneamide], a plant-derived vanilloid agonist, and capsazepine (N-[2-(4-chlorophenyl)ethyl]-1,3,4,5-tetrahydro-7,8-dihydroxy-2H-2-benzazepine-2-carbothioamide), a vanilloid antagonist, had no such effect (Ki>100 μM). These results indicate that, although less potent than AM404 (Ki 2.1±0.2 μM), olvanil may reduce **anandamide** clearance at concns. similar to those needed for vanilloid receptor activation.

IT **404-86-4**, Capsaicin **58493-49-5**, Olvanil
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**anandamide transport inhibition** by vanilloid agonist olvanil)

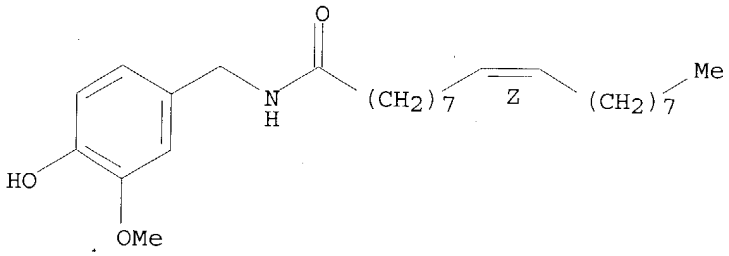
RN 404-86-4 HCAPLUS
CN 6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 58493-49-5 HCAPLUS
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z)- (9CI) (CA INDEX NAME)

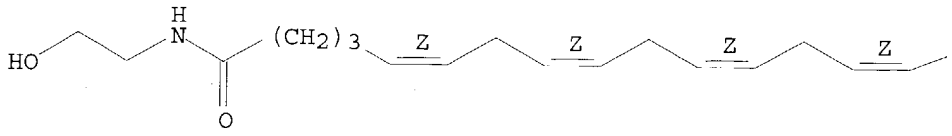
Double bond geometry as shown.



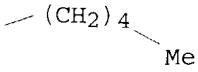
IT **94421-68-8, Anandamide**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(**anandamide transport inhibition** by vanilloid agonist olvanil)
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS

RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

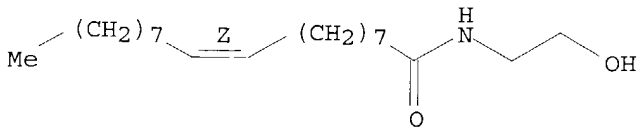
L45 ANSWER 5 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:698015 HCAPLUS
DOCUMENT NUMBER: 130:76092
TITLE: Interactions between synthetic vanilloids and the endogenous cannabinoid system
AUTHOR(S): Di Marzo, Vincenzo; Bisogno, Tiziana; Melck, Dominique; Ross, Ruth; Brockie, Heather; Stevenson, Lesley; Pertwee, Roger; De Petrocellis, Luciano
CORPORATE SOURCE: Istituto per la Chimica di Molecole di Interesse Biologico, CNR, Arco Felice, 80072, Italy
SOURCE: FEBS Letters (**1998**), 436(3), 449-454
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The chemical similarity between some synthetic agonists of vanilloid receptors, such as olvanil (N-vanillyl-cis-9-octadecenoamide), and the 'endocannabinoid' **anandamide** (arachidonoyl-ethanolamide, AEA), suggests possible interactions between the cannabinoid and vanilloid signalling systems. Here the authors report that olvanil is a stable and potent **inhibitor** of AEA facilitated **transport** into rat basophilic leukemia (RBL-2H3) cells. Olvanil blocked both the uptake and the hydrolysis of [14C]AEA by intact RBL-2H3 cells (IC50 = 9 μM), while capsaicin and pseudocapsaicin (N-vanillyl-nonanamide) were much less active. Olvanil was more potent than previously reported **inhibitors** of AEA facilitated **transport**, i.e. phloretin (IC50 = 80 μM), AM404 (12.9%, **inhibition** at 10 μM) or oleoylethanolamide (27.5% **inhibition** at 10 μM). Olvanil was a poor **inhibitor** of [14C]AEA hydrolysis by RBL-2H3 and N18TG2 cell membranes, suggesting that the **inhibitory** effect on [14C]AEA breakdown observed in intact cells was due to **inhibition** of [14C]AEA uptake. Olvanil was stable to enzymic hydrolysis, and (i) displaced the binding of high affinity cannabinoid receptor ligands to membrane prepns. from N18TG2 cells and guinea pig forebrain (Ki = 1.64-7.08 μM), but not from cells expressing the CB2 cannabinoid receptor subtype; (ii) **inhibited** forskolin-induced cAMP formation in intact N18TG2 cells (IC50 = 1.60 μM), this effect being reversed by the selective CB1 antagonist SR141716A. Pseudocapsaicin, but not capsaicin, also selectively bound to CB1 receptor-containing membranes. These data suggest that some of the analgesic actions of olvanil may be due to its interactions with the endogenous cannabinoid system, and may lead to the design of a novel class of cannabimimetics with potential therapeutic applications as analgesics.

IT **111-58-0** **404-86-4**, Capsaicin **58493-49-5**, Olvanil **94421-68-8**, Anandamide
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(interactions between synthetic vanilloids and the endogenous cannabinoid system)

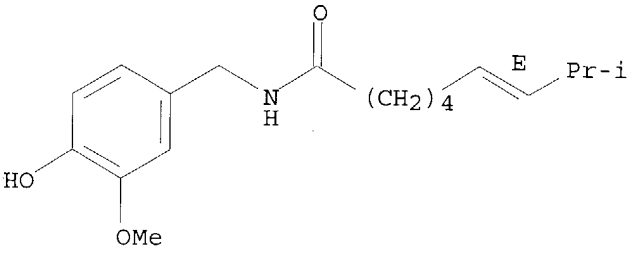
RN 111-58-0 HCAPLUS
CN 9-Octadecenamide, N-(2-hydroxyethyl)-, (9Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



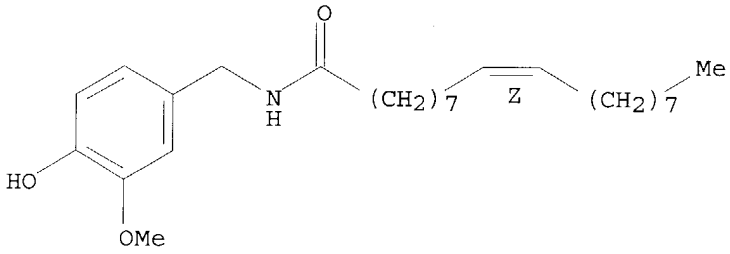
RN 404-86-4 HCAPLUS
CN 6-Nonenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-8-methyl-, (6E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 58493-49-5 HCAPLUS
CN 9-Octadecenamide, N-[(4-hydroxy-3-methoxyphenyl)methyl]-, (9Z)- (9CI) (CA INDEX NAME)

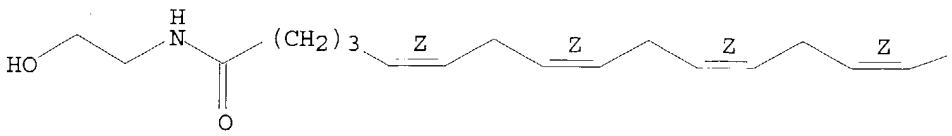
Double bond geometry as shown.



RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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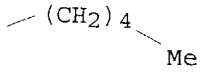


REFERENCE COUNT: 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 6 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:808015 HCAPLUS
DOCUMENT NUMBER: 128:136686
TITLE: Inhibition of intestinal motility by anandamide, an endogenous cannabinoid
AUTHOR(S): Calignano, Antonio; La Rana, Giovanna; Makriyannis, Alexandros; Lin, Sun Y.; Beltramo, Massimiliano; Piomelli, Daniele
CORPORATE SOURCE: Department of Experimental Pharmacology, University of Naples, Naples 80123, Italy
SOURCE: European Journal of Pharmacology (**1997**), 340(2/3), R7-R8
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English

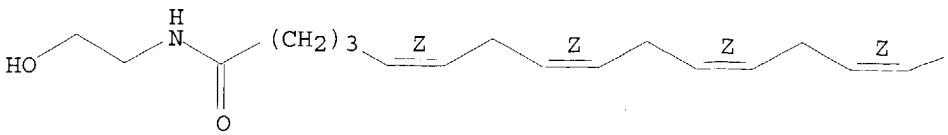
AB The endogenous cannabinoid ligand **anandamide** (arachidonylethanolamide) **inhibited** the intestinal passage of a charcoal meal when administered s.c. in mice at doses ranging from 0.1 to 50 mg/kg. This effect was prevented by the cannabinoid CB1 receptor antagonist SR141716A [N-(piperidin-1-yl)-5-(4-chlorophenyl)-1-(2,4-dichlorophenyl)-4-methyl-1H-pyrazole-3-carboxamide·HCl] (1 mg/kg s.c.), but it was not affected by the **anandamide transport inhibitor**, N-(4-hydroxyphenyl) arachidonylethanolamide (AM404) (50 mg/kg, s.c.). The results indicate that **anandamide** modulates intestinal motility in mice by activating cannabinoid CB1 receptors. They also suggest that **anandamide transport**, which was previously shown to participate in terminating neural and vascular responses to **anandamide**, does not contribute to **anandamide** inactivation in intestinal tissue.

IT **94421-68-8**, Anandamide
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(intestinal motility inhibition by anandamide mediation by cannabinoid receptors)

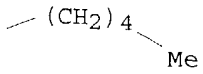
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



REFERENCE COUNT: 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L45 ANSWER 7 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:646587 HCAPLUS
DOCUMENT NUMBER: 127:329390
TITLE: Potentiation of **anandamide** hypotension by the **transport inhibitor**, AM404
AUTHOR(S): Calignano, Antonio; La Rana, Giovanna; Beltramo, Massimiliano; Makriyannis, Alexandros; Piomelli, Daniele
CORPORATE SOURCE: Department of Experimental Pharmacology, University of Naples, Naples, 80123, Italy
SOURCE: European Journal of Pharmacology (**1997**), 337(1), R1-R2
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

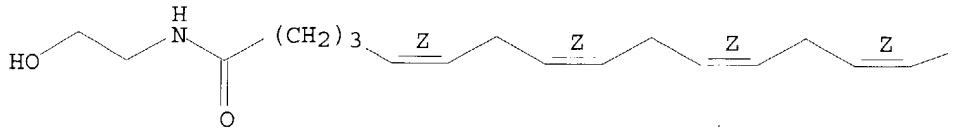
AB The putative endogenous cannabinoid, **anandamide** (0.2-2 mg/kg i.v.), decreased systemic blood pressure dose-dependently in anesthetized guinea pigs. These effects were prevented by the CB1 cannabinoid receptor antagonist SR141716A [N-(piperidin-1-yl)-5-(4-chlorophenyl)-1-(2,4-dichlorophenyl)-4-methyl-1H-pyrazole-3-carboxamide·HCl] at the dose of 0.2 mg/kg i.v. The vasodepressor responses to **anandamide** were significantly potentiated and prolonged by a novel **inhibitor** of carrier-mediated **anandamide transport**, N-(4-hydroxyphenyl) arachidonylethanolamide (AM404) (10 mg/kg, i.v.). These results suggest that **anandamide transport** participates in terminating the vascular actions of **anandamide.**

IT **94421-68-8**, **Anandamide**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(potentiation of **anandamide** hypotension by **transport inhibitor**, AM404)

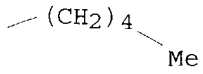
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



L45 ANSWER 8 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:550217 HCAPLUS
DOCUMENT NUMBER: 127:246072
TITLE: Functional role of high-affinity **anandamide transport**, as revealed by selective **inhibition**
AUTHOR(S): Beltramo, M.; Stella, N.; Calignano, A.; Lin, S. Y.; Makriyannis, A.; Piomelli, D.
CORPORATE SOURCE: The Neurosciences Inst., San Diego, CA, 92121, USA
SOURCE: Science (Washington, D. C.) (**1997**), 277(5329), 1094-1097
CODEN: SCIEAS; ISSN: 0036-8075
PUBLISHER: American Association for the Advancement of Science
DOCUMENT TYPE: Journal
LANGUAGE: English

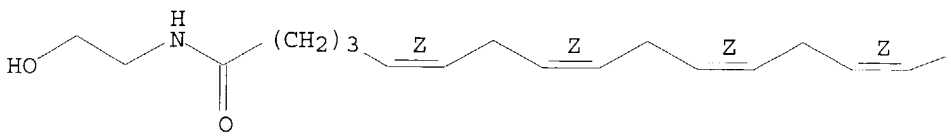
AB **Anandamide**, an endogenous ligand for central cannabinoid receptors, is released from neurons on depolarization and rapidly inactivated. **Anandamide** inactivation is not completely understood, but it may occur by **transport** into cells or by enzymic hydrolysis. The compound N-(4-hydroxyphenyl)arachidonylamide (AM404) was shown to **inhibit** high-affinity **anandamide** accumulation in rat neurons and astrocytes in vitro, an indication that this accumulation resulted form carrier-mediated **transport.** Although AM404 did not activate cannabinoid receptors or **inhibit anandamide** hydrolysis, it enhanced receptor-mediated **anandamide** responses in vitro and in vivo. The data indicate that carrier-mediated **transport** may be essential for termination of the biol. effects of **anandamide**, and may represent a potential drug target.

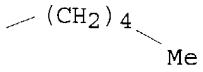
IT **94421-68-8**, Anandamide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(carrier-mediated transport of anandamide)

RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A





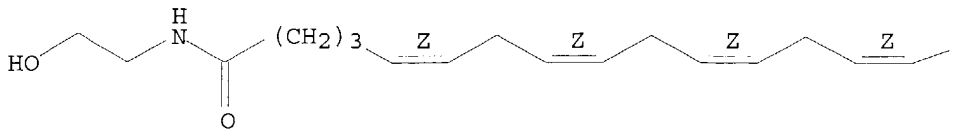
L45 ANSWER 9 OF 9 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1997:495779 HCAPLUS
DOCUMENT NUMBER: 127:188622
TITLE: Accumulation of N-arachidonoylethanolamine (anandamide) into cerebellar granule cells occurs via facilitated diffusion
AUTHOR(S): Hillard, Cecilia J.; Edgemond, William S.; Jarrahian, Abbas; Campbell, William B.
CORPORATE SOURCE: Department of Pharmacology and Toxicology, Medical College of Wisconsin, Milwaukee, WI, 53226, USA
SOURCE: Journal of Neurochemistry (**1997**), 69(2), 631-638
CODEN: JONRA9; ISSN: 0022-3042
PUBLISHER: Lippincott-Raven
DOCUMENT TYPE: Journal
LANGUAGE: English

AB N-Arachidonoylethanolamine (**anandamide**, AEA) is a putative endogenous ligand of the cannabinoid receptor. Intact cerebellar granule neurons in primary culture rapidly accumulate AEA. [3H]AEA accumulation by cerebellar granule cells is dependent on incubation time (t1/2 of 2.6 ± 0.8 min at 37°C) and temperature The accumulation of AEA is saturable and has an apparent Km of 41 ± 15 μM and a Vmax of 0.61 ± 0.04 nmol/min/106 cells. [3H]AEA,accumulation by cerebellar granule cells is significantly reduced by 200 μM phloretin (57.4 ± 4% of control) in a noncompetitive manner. [3H]AEA accumulation is not **inhibited** by either ouabain or removal of extracellular sodium. [3H]AEA accumulation is fairly selective for AEA among other naturally occurring N-acylethanolamines; only N-oleoylethanolamine significantly **inhibited** [3H]AEA accumulation at a concentration of 10 μM. The ethanolamides of palmitic acid and linolenic acid were inactive at 10 μM. N-Arachidonoylbenzylamine and N-arachidonoylpropylamine, but not arachidonic acid, 15-hydroxy-AEA, or 12-hydroxy-AEA, compete for AEA accumulation. When cells are preloaded with [3H]AEA, temperature-dependent efflux occurs with a half-life of 1.9 ± 1.0 min. Phloretin does not **inhibit** [3H]AEA efflux from cells. These results suggest that AEA is accumulated by cerebellar granule cells by a protein-mediated **transport** process that has the characteristics of facilitated diffusion.

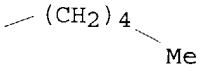
IT **94421-68-8**, Anandamide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(accumulation of N-arachidonoylethanolamine into cerebellar granule cells occurs via facilitated diffusion)
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B



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R 2051 OR 2043
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L39        1358 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L36 OR ?ANANDAMID?
L43          70 SEA FILE=HCAPLUS ABB=ON  PLU=ON  INHIBIT?(L)TRANSPORT(L)L39
L44          62 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L43 AND L38
L45           9 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L44 AND PD=<JUNE 9, 1999
L46           1 SEA FILE=REGISTRY ABB=ON  PLU=ON  ARACHIDONOYLETHAN/BI
L47             SEL  PLU=ON  L46 1- CHEM :       7 TERMS
L48        1286 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L47
L49        1289 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L48 OR ?ARACHIDONOYLETHAN?
L50          98 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L49(L)INHIBIT?(L)TRANSPOR?
L51          86 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L50 AND L38
L52           3 SEA FILE=HCAPLUS ABB=ON  PLU=ON  (L51 AND PD=<JUNE 9, 1999)
                NOT L45


=>
=> d ibib abs hitstr l52 1-3

L52  ANSWER 1 OF 3  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:         1999:735737  HCAPLUS
DOCUMENT NUMBER:          132:62244
TITLE:                    The endothelial component of cannabinoid-induced
                          relaxation in rabbit mesenteric artery depends on gap
                          junctional communication
AUTHOR(S):                Chaytor, A. T.; Martin, P. E. M.; Evans, W. H.;
                          Randall, M. D.; Griffith, T. M.
CORPORATE SOURCE:         Departments of Diagnostic Radiology and Cardiovascular
                          Sciences Research Group, University of Wales College
                          of Medicine, Cardiff, CF4 4XN, UK
SOURCE:                   Journal of Physiology (Cambridge, United Kingdom) (
                          1999), 520(2), 539-550
                          CODEN: JPHYA7; ISSN: 0022-3751
PUBLISHER:                Cambridge University Press
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB    1. The authors have shown that the endocannabinoid anandamide
      and its stable analog methanandamide relax rings of rabbit superior
      mesenteric artery through endothelium-dependent and -independent
      mechanisms that are unaffected by blockade of NO synthase and
      cyclooxygenase.  2. The endothelium-dependent component of the responses
      was attenuated by the gap junction inhibitor
      18α-glycyrrhetinic acid (18α-GA; 50 μM), and a synthetic
      connexin-mimetic peptide homologous to the extracellular Gap 27 sequence
      of connexin 43 (43Gap 27, SRPTEKTIFII; 300 μM).  By contrast, the
      corresponding connexin 40 peptide (40Gap 27, SRPTEKNVFIV) was inactive.
      3. The cannabinoid CB1 receptor antagonist SR141716A (10 μM) also
      attenuated endothelium-dependent relaxations but this inhibition
      was not observed with the CB1 receptor antagonist LY320135 (10 μM).
      Furthermore, SR141716A mimicked the effects of 43Gap 27 peptide in
      blocking Lucifer Yellow dye transfer between coupled COS-7 cells (a monkey
      fibroblast cell line), whereas LY320135 was without effect, thus
      suggesting that the action of SR141716A was directly attributable to
      effects on gap junctions.  4. The endothelium-dependent component of
      cannabinoid-induced relaxation was also attenuated by AM404 (10 μM), an
      inhibitor of the high-affinity anandamide
      transporter, which was without effect on dye transfer.  5. Taken
      together, the findings suggest that cannabinoids derived from arachidonic
      acid gain access to the endothelial cytosol via a transporter
      mechanism and subsequently stimulate relaxation by promoting diffusion of
```

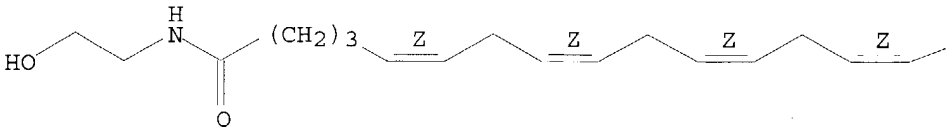
an endothelium-derived hyperpolarizing factor to adjacent smooth muscle cells via gap junctions. 6. Relaxations of endothelium-denuded prepns. to **anandamide** and methanandamide were unaffected by 43Gap 27 peptide, 18α-GA, SR141716A, AM404 and indomethacin and their genesis remains to be established.

IT **94421-68-8**, Anandamide **150314-39-9**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(endothelial component of cannabinoid-induced relaxation in rabbit mesenteric artery depends on gap junctional communication and not on CB1 receptors)

RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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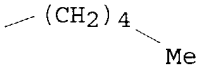
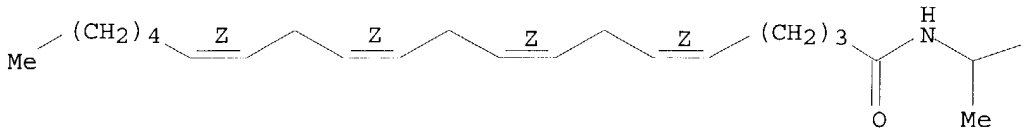


RN 150314-39-9 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxy-1-methylethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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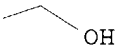


REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

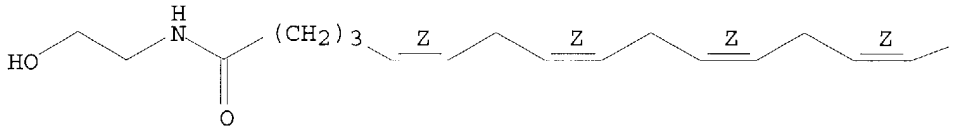
L52 ANSWER 2 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:262822 HCAPLUS
DOCUMENT NUMBER: 131:71734
TITLE: Anandamide activates human platelets through a pathway independent of the arachidonate cascade

AUTHOR(S): Maccarrone, Mauro; Bari, Monica; Menichelli, Adriana; Del Principe, Domenico; Finazzi Agro, Alessandro
CORPORATE SOURCE: Department of Experimental Medicine and Biochemical Sciences, University of Rome Tor Vergata, Rome, I-00133, Italy
SOURCE: FEBS Letters (**1999**), 447(2,3), 277-282
CODEN: FEBLAL; ISSN: 0014-5793
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Anandamide** (**arachidonoylethanolamide**, AnNH) is shown to activate human platelets, a process which was not **inhibited** by acetylsalicylic acid (aspirin). Unlike AnNH, hydroperoxides generated thereof by lipoxygenase activity, and the congener (13-hydroxy)linoleoylethanolamide, were unable to activate platelets, though they counteracted AnNH-mediated stimulation. On the other hand, palmitoylethanolamide neither activated human platelets nor blocked the AnNH effects. AnNH inactivation by human platelets was afforded by a high-affinity **transporter**, which was activated by nitric oxide-donors up to 225% of the control. The internalized AnNH could thus be hydrolyzed by a fatty acid amide hydrolase (FAAH), characterized here for the first time.

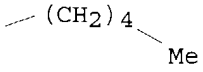
IT **94421-68-8**, Anandamide
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(anandamide activates human platelets through a pathway independent of arachidonate cascade)
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A

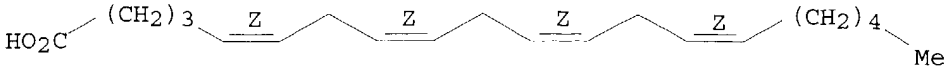


PAGE 1-B



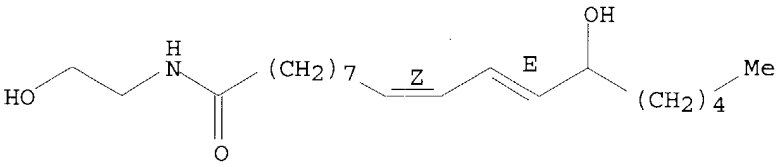
IT **506-32-1**, Arachidonic acid **219931-42-7**
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(anandamide activates human platelets through a pathway independent of arachidonate cascade)
RN 506-32-1 HCAPLUS
CN 5,8,11,14-Eicosatetraenoic acid, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 219931-42-7 HCAPLUS
CN 9,11-Octadecadienamide, 13-hydroxy-N-(2-hydroxyethyl)-, (9Z,11E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



REFERENCE COUNT: 40 THERE ARE 40 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L52 ANSWER 3 OF 3 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:783697 HCAPLUS
DOCUMENT NUMBER: 130:122662
TITLE: Anandamide hydrolysis by human cells in culture and brain
AUTHOR(S): Maccarrone, Mauro; Van Der Stelt, Marcelis; Rossi, Antonello; Veldink, Gerrit A.; Vliegenthart, Johannes F. G.; Agro, Alessandro Finazzi
CORPORATE SOURCE: Department of Experimental Medicine and Biochemical Sciences, University of Rome Tor Vergata, Rome, I-00133, Italy
SOURCE: Journal of Biological Chemistry (**1998**), 273(48), 32332-32339
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Anandamide** (**arachidonylethanolamide**; AnNH) has important neuromodulatory and immunomodulatory activities. This lipid is rapidly taken up and hydrolyzed to arachidonate and ethanolamine in many organisms. As yet, AnNH inactivation has not been studied in humans. Here, a human brain fatty-acid amide hydrolase (FAAH) has been characterized as a single protein of 67 kDa with a pI of 7.6, showing apparent Km and Vmax values for AnNH of 2.0 ± 0.2 μM and 800 ± 75 pmol·min-1·mg of protein-1, resp. The optimum pH and temperature for AnNH hydrolysis were 9.0 and 37 °C, resp., and the activation energy of the reaction was 43.5 ± 4.5 kJ·mol-1. Hydro(pero)xides derived from AnNH or its linoleoyl analogs by lipoxygenase action were competitive **inhibitors** of human brain FAAH, with apparent Ki values in the low micromolar range. One of these compds., linoleoylethanolamide is the first natural **inhibitor** (Ki = 9.0 ± 0.9 μM) of FAAH as yet discovered. An FAAH activity sharing several biochem. properties with the human brain enzyme was demonstrated in human neuroblastoma CHP100 and lymphoma U937 cells. Both cell lines have a high affinity **transporter** for AnNH, which had apparent Km and Vmax values for AnNH of 0.20 ± 0.02 μM and 30 ± 3 pmol·min-1·mg of protein-1 (CHP100 cells) and 0.13 ± 0.01 μM and 140 ± 15 pmol·min-1·mg of protein-1 (U937

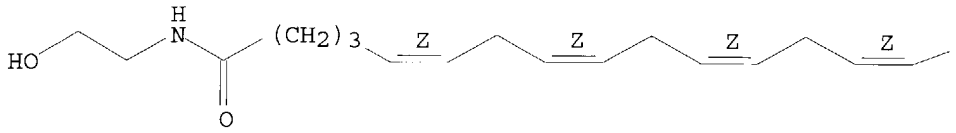
cells), resp. The AnNH carrier of both cell lines was activated up to 170% of the control by nitric oxide.

IT **94421-68-8**, Anandamide
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(anandamide hydrolysis by human cells in culture and brain)

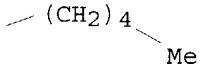
RN 94421-68-8 HCAPLUS
CN 5,8,11,14-Eicosatetraenamide, N-(2-hydroxyethyl)-, (5Z,8Z,11Z,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



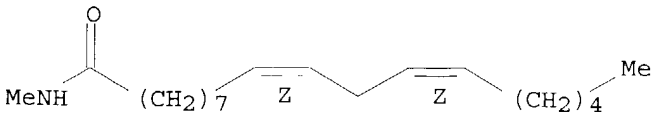
PAGE 1-B



IT **3140-44-1 3999-01-7 68171-52-8**
171627-25-1 171756-49-3 219931-40-5
219931-41-6 219931-42-7 219931-43-8
219931-44-9
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(in brain fatty-acid amide hydrolase inhibition and anandamide hydrolysis by human cells in culture and brain)

RN 3140-44-1 HCAPLUS
CN 9,12-Octadecadienamide, N-methyl-, (9Z,12Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 3999-01-7 HCAPLUS
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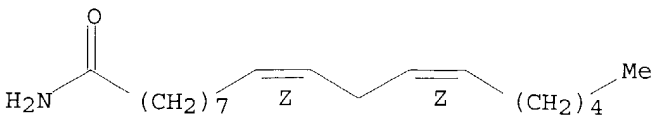
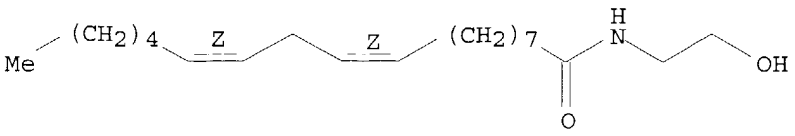
Double bond geometry as shown.



RN 68171-52-8 HCAPLUS
CN 9,12-Octadecadienamide, N-(2-hydroxyethyl)-, (9Z,12Z)- (9CI) (CA INDEX NAME)

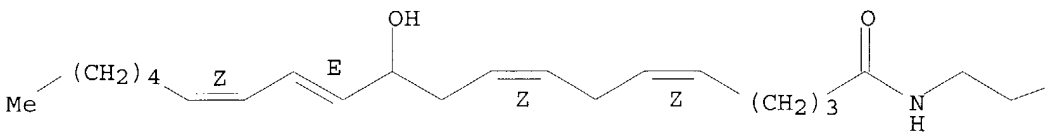
Double bond geometry as shown.



RN 171627-25-1 HCAPLUS
CN 5,8,12,14-Eicosatetraenamide, 11-hydroxy-N-(2-hydroxyethyl)-, (5Z,8Z,12E,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A

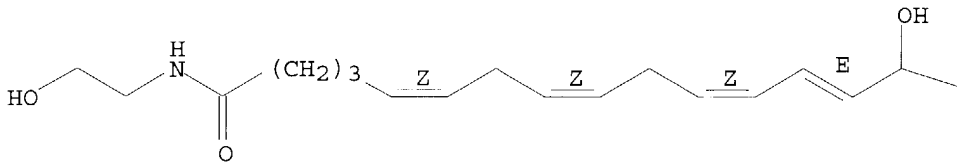


PAGE 1-B

RN 171756-49-3 HCAPLUS
CN 5,8,11,13-Eicosatetraenamide, 15-hydroxy-N-(2-hydroxyethyl)-, (5Z,8Z,11Z,13E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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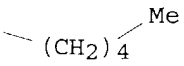
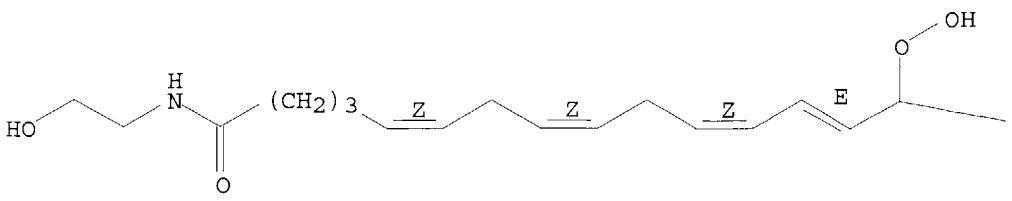


RN 219931-40-5 HCAPLUS
CN 5,8,11,13-Eicosatetraenamide, 15-hydroperoxy-N-(2-hydroxyethyl)-, (5Z,8Z,11Z,13E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



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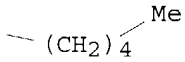
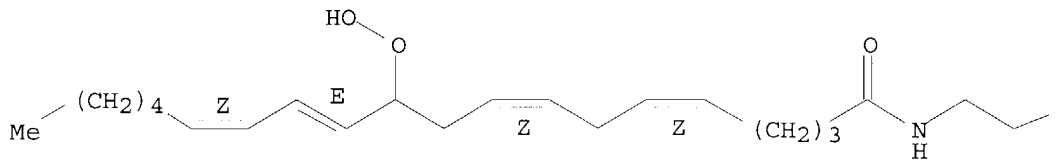


RN 219931-41-6 HCAPLUS
CN 5,8,12,14-Eicosatetraenamide, 11-hydroperoxy-N-(2-hydroxyethyl)-, (5Z,8Z,12E,14Z)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.

PAGE 1-A



PAGE 1-B

RN 219931-42-7 HCAPLUS
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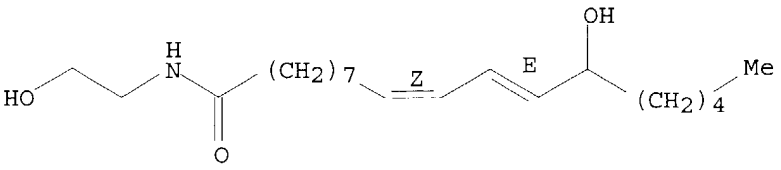
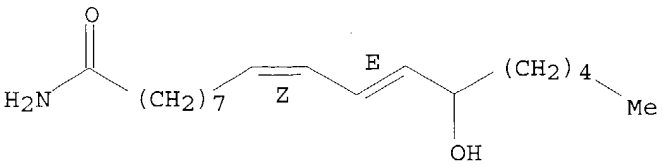
Double bond geometry as shown.



RN 219931-43-8 HCAPLUS
CN 9,11-Octadecadienamide, 13-hydroxy-, (9Z,11E)- (9CI) (CA INDEX NAME)

Double bond geometry as shown.



RN 219931-44-9 HCAPLUS
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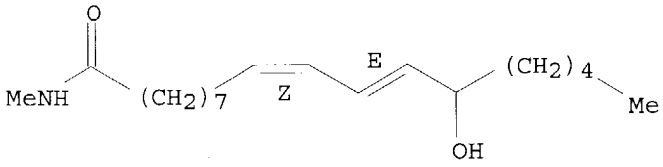
Double bond geometry as shown.



REFERENCE COUNT: 46 THERE ARE 46 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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